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GENETIC SUSCEPTIBILITY TESTING IN SMOKING-CESSATION TREATMENT: ONE-YEAR OUTCOMES OF A RANDOMIZED TRIAL

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Abstract — This study evaluated the long-term impact of genetic susceptibility biomarker feedback on smoking behavior change and symptoms of depression in 426 male and female smokers. Smokers were randomized to one of three smoking-cessation interventions: minimal contact quit-smoking counseling (QSC), QSC + exposure biomarker feedback (EBF), and QSC + EBF + biomarker feedback about genetic susceptibility to lung cancer (SBF). The logistic regression model for quit attempt revealed a significant main effect for treatment such that participants in the SBF group were more than two times more likely to make a quit attempt than participants in the QSC group. There was not a significant difference between EBF and QSC participants. The results also revealed a significant effect for baseline stage of change. Those smokers in the preparation stage at baseline were more than three times more likely to make a quit attempt over the 12 months following treatment. The models for 30-day cessation and follow-up smoking rate revealed no significant main or interacting effects for treatment. A repeated measures analysis of variance revealed a significant main effect for time, indicating that an initial increase in depression in the genetic susceptibility group was not maintained over time. Genetic susceptibility feedback has the intended effects on motivation to quit, but it may need to be delivered within a more intensive smoking-cessation treatment for the heightened motivation to translate into smoking cessation. © 1997 Elsevier Science Ltd

Approximately 48 million adults in the United States smoke cigarettes ("Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion, CDC" 1996). In the past decade, there has been very little change in smoking prevalence among adults (National Center for Health Statistics, 1996), and the results of large-scale population-based smoking trials have been disappointing (Shiffman, 1993). More individualized smoking-cessation treatments may be needed to enhance motivation to quit among adult smokers (West & Grunberg, 1991).

We reported previously on the immediate and short-term (2-month) impact of individualized motivational feedback about genetic susceptibility to lung cancer (Lerman et al., 1997) The results of this randomized trial indicated significant positive effects of genetic susceptibility feedback, compared to standard counseling and counseling plus carbon monoxide (CO) feedback, on perceived risk, perceived quitting benefits, and fear arousal. However, between-groups differences in short-term cessation rates were not found. Further, there was evidence for small but significant increases in depression in the genetic susceptibility group compared to the other conditions. This article re-

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ports on the long-term (12-month) impact of genetic susceptibility feedback on smoking behavior change and on symptoms of depression. In addition, we evaluated two potential moderators of treatment effects—stage of change and self-efficacy.

The transtheoretical model defines smoking behavior change as a set of stages through which smokers progress: (a) *precontemplation*, or not intending to quit; (b) *contemplation*, or seriously considering quitting in the next 6 months; (3) *preparation*, or planning to quit in the next month and making a recent quit attempt; (d) *action*, or recently quit; and (e) *maintenance*, or maintaining smoking abstinence (Prochaska et al., 1994). Smokers who are in the preparation stage are significantly more likely to attempt to quit and to abstain from smoking in the subsequent months than are smokers in the precontemplation or contemplation stages (DiClemente et al., 1991; Gritz, Berman, Bastani, & Wu, 1992). Although stage of change is a strong predictor of future smoking behavior change, little is known about whether stage of change moderates the impact of smoking-cessation treatments. We predicted that smokers in the preparation stage of change would be more responsive to genetic susceptibility feedback than smokers in the precontemplation and contemplation stages of change.

Stage of readiness to change smoking behavior has also been found to relate to self-efficacy (DiClemente, 1986; DiClemente et al., 1991; Owen, Wakefield, Roberts, & Esterman, 1992). Quitting self-efficacy has been linked to initial cessation rates and to relapse following smoking cessation (Brandon, Tiffany, Obremski, & Baker, 1990; DiClemente, Prochaska, & Gibertini, 1985; Garvey, Bliss, Hitchcock, Heinold, & Rosner, 1992). In addition, smokers with high levels of self-efficacy appear to respond better to social support smoking-cessation treatments, whereas those low in self-efficacy respond better to self-control smoking-cessation treatments (Digiusto & Bird, 1995). Given the motivational component of social support treatments, we predicted that smokers with high levels of self-efficacy would also respond better to motivational feedback about genetic susceptibility than smokers with low levels of self-efficacy.

METHOD

Participants

The participants were 426 male and female smokers between the ages of 18 and 75 who responded to advertisements for a free smoking-cessation study involving a minimal contact intervention. Eligible smokers were those currently smoking at least five cigarettes a day for at least a year. Smokers who were pregnant, currently undergoing drug and/or alcohol treatment, had a psychiatric disorder that precluded informed consent, or a history of cancer were excluded. A subset of this sample was used as the basis for a previous report on depression and nicotine dependence (Lerman et al., 1996). The present sample was used as the basis for a previous report on the immediate and short-term effects of providing genetic susceptibility feedback on smoking-related cognitions, emotions, and behavior (Lerman et al., 1997).

Design and procedures

The study design, procedures, and interventions are described in depth in our previous report (Lerman et al., 1997). Briefly, persons responding to newspaper advertisements received a short telephone interview to determine eligibility. If eligible, they received a brief description of the study and participation requirements. During an initial visit to the Smoking Clinic, all subjects completed a set of self-report questionnaires assessing demographics, smoking history, quitting readiness, quitting confidence, and de-

pressive symptoms. The benefits, risks, and limitations (e.g., genotyping, test uncertainty, loss of privacy) of participation were explained verbally and in the written consent form (American Society of Human Genetics, 1994; Rothstein, 1990; Wilfond & Fost, 1990). Subjects who consented to participate provided a 40-cc blood sample.

Two to three weeks after the initial visit to the Smoking Clinic, subjects met with a trained health educator who delivered one of the three smoking-cessation interventions. This was determined by random assignment. Two-month and 12-month follow-up telephone interviews were conducted to assess short-term changes and long-term changes in smoking practices. This article reports on 12-month follow-up data.

Quit-smoking consultation (QSC). Smokers randomized to this condition received a standardized 60-min individual smoking-cessation consultation. The QSC condition was modeled after the intervention found effective by Orleans, Rotberg, Quade, and Lees (1990) and was based on the *Free & Clear* guide evaluated in a large, randomized self-help quite-smoking trial (Orleans et al., 1991). Counseling included a motivational review of smoking and quitting history, the development of a personal quitting plan, a discussion of potential for reductions in risks of lung cancer and other smoking-related diseases following cessation, directions for following a nicotine-fading protocol, and brief advice on gaining support for quitting efforts.

Exposure biomarker feedback (EBF). Smokers randomized to this condition received the complete QSC intervention plus an additional 10-min motivational intervention delivered prior to the QSC. The motivational component involved the assessment of CO levels via a BreathCo carbon monoxide monitor (Vitalograph, Inc., Lenexa, KS). The CO feedback protocol, modeled after that used by Risser and Belcher (1990), was designed to highlight the deleterious effects of smokers' tobacco exposure relative to the exposures of ex-smokers and nonsmokers.

Susceptibility biomarker feedback (SBF). Smokers randomized to this condition received the complete EBS + QSC intervention (noted before) plus an additional 10-min motivational intervention delivered prior to the QSC. The motivational component involved feedback of the results of genotyping for CYP2D6. The CYP2D6 gene codes for an enzyme involved in the metabolism of tobacco carcinogens. It is the most widely studied marker of lung cancer risk. Persons who have genotypes enabling extensive metabolism have a twofold to fourfold risk of developing lung cancer (Amos, Caporaso, & Weston, 1992). CYP2D6 genotype was analyzed from the blood samples of subjects in the SBF group using a polymerase chain reaction assay.

Standardized feedback messages emphasized a smokers' susceptibility to lung cancer relative to that of other smokers. Thus, subjects in this condition received personalized feedback, not only of their exposure to tobacco (i.e., CO levels) but also of their individual susceptibility to this exposure (i.e., CYP2D6 genotype). Susceptibility feedback included a discussion of the role of genes in metabolism (activation) of carcinogens in tobacco and how extensive metabolizers are more susceptible to lung cancer than poor metabolizers, a discussion of the uncertainty inherent in estimates of cancer risk and the need for further studies to refine genetic markers, and personal feedback of the results of CYP2D6 genotyping.

Measures

Control variables.

Background and smoking history assessment. A detailed smoking history questionnaire was administered at baseline to collect the following data: demographic characteristics (age, gender, ethnicity, marital status, and education), age at smoking initiation, longest prior abstinence period, and current smoking rate.

Dependent variables.

Smoking behavior. At the 12-month follow-up, participants were asked to respond to the following dichotomous (yes, no) items: (a) "Since your counseling session about 12 months ago, have you tried to quit smoking?" and (b) "During the past 30 days, have you smoked a cigarette, even a puff?" Those participants who reported continued smoking were asked about their current smoking rate.

Center for epidemiologic studies depression scale (CES-D). The CES-D is a 20-item Likert-style scale used to assess depressive symptoms. The CES-D has high internal consistency (r = .85–.90) and has been shown to correlate with clinical ratings of the severity of depression (Radloff, 1977). In our sample, the Cronbach's alpha estimate of internal consistency was .90 for baseline CES-D.

Predictor/moderator variables.

Quitting readiness. Readiness to quit smoking, or stage of change, was measured at baseline and at the 12-month follow-up using a single forced-choice item based on well-validated stage measures (DiClemente et al., 1991; Prochaska et al., 1994). Subjects were classified into one of three prequitting stages: (a) precontemplation—those not seriously considering quitting in the next 6 months; (b) contemplation—those seriously considering quitting in the next 6 months; and (c) preparation—those planning to quit within the next 30 days and who also reported a quit attempt in the past 12 months. Those smokers planning to quit in the next 30 days, but who did not make a quit attempt in the past 12 months, were classified as contemplators (Prochaska et al., 1994). Given the small number of participants who were in the precontemplation stage (n = 11), a dichotomous variable for stage of change was used (e.g., preparation vs. else).

Self-efficacy (quitting confidence). A Likert-style item was used to measure self-efficacy or confidence. This item was adapted from items used in smoking intervention outcomes research (National Cancer Institute; 1986). The self-efficacy item asked, "How confident are you that you could quit smoking for good?" (0 = *not at all*, 1 = *a little*, = *somewhat*, 3 = *very much*, 4 = *extremely*). This single-item measure has predicted smoking cessation in several prospective studies of self-help treatments (Orleans et al., 1991; Rimer & Orleans, 1994). Due to low cell frequencies for some response options, self-efficacy was dichotomized (e.g., "very much/extremely" vs. "not at all/a little/somewhat").

RESULTS

Response rates

A total of 1,252 individuals responded to the newspaper advertisement and completed the telephone eligibility screening. Of these, 1,104 (88%) met the eligibility criteria and were invited to participate. After receiving a description of the study requirements, 952 individuals (86%) agreed to participate. Of those who agreed, 550 (58%) completed both visits (i.e., a preliminary visit to complete self-report question-

naires and a smoking intervention visit). Those individuals who declined participation (n = 152) after receiving a description of the study requirements did not differ from those individuals who agreed to participate in the study (n = 952) on the demographic or smoking history variables assessed by the eligibility screen (e.g., age, smoking rate, years smoked). Participants who completed both visits (n = 550), however, did differ from those who withdrew before or after the initial visit (n = 402) on demographic and smoking history variables. Those who completed both visits tended to be older (44 vs. 40 years old; $t(912) = -5.60, p = .0001$), smoked more cigarettes per day (23 vs. 21; $t(943) = -3.49, p = .0005$), and smoked longer (25 vs. 21 years; $t(914) = -5.42, p = .0001$). Participants who completed the 12-month follow-up—77% of participants who completed both visits or 426 of 550—did not differ from participants who were lost to follow-up on any baseline smoking history variable; however, women were more likely than men to complete the 12-month follow-up—87% versus 78%; $\chi^2(1, N = 426) = 8.25, p = .004$. There were no significant differences between treatment groups with respect to loss to follow-up; χ^2 (2, N = 550) = 0.54, p = .77.

Characteristics of the study sample

As shown in Table 1, the three treatment groups did not differ significantly at baseline on any of the demographic or smoking history variables. The sample was predominantly White (83.9%), highly educated, and over half of the participants were women (62.8%). The participants tended to be heavy smokers, consuming more than one pack of cigarettes a day, and tended to be moderately to heavily dependent on nicotine (average Fagerstrom Test of Nicotine Dependence score = 5.4, SD = 2.3, range = 0–10). The average CO level in the sample was 30 ppm ± 15 (measured in EBF and SBF groups only). The average CES-D score at baseline was 14.5 (SD = 9.9), suggesting a moderate level of depressive symptomatology. Thirty-four percent of participants in the QSC group, 32% of participants in the EBF group, and 40% of participants in the SBF group reported feeling very much/extremely confident in their ability to quit smoking. At baseline, 38% of QSC participants were in the preparation stage of change, 35% of EBF participants were in the preparation stage, and 40% of SBF participants were in the preparation stage. There were no significant between-groups differences in baseline depression, quitting self-efficacy, or stage of change.

Table 1. Baseline characteristics of study population by treatment group

	Treatment group								
	QSC (n = 137)			EBF (n = 156)			SBF (n = 133)		
Variable	M	SD	%	M	SD	%	M	SD	%
Demographic									
Age	44.8	11.2		44.1	12.7		43.2	11.2	
Gender (% female)			65.2			64.5			58.3
Education (>high school)			72.8			83.8			90.0
Ethnicity (non-White)			14.5			16.8			17.1
Employment (full time)			54.7			59.5			65.1
Marital status (married)			46.3			37.0			43.8
Smoking practices									
Age at initiation	16.6	3.7		16.5	3.1		16.4	3.7	
Cigarettes/day	21.7	9.9		23.6	12.0		22.7	10.2	
Nicotine dependence	5.5	2.3		5.4	2.3		5.3	2.4	

Note. QSC = quit-smoking counseling; EBF = exposure biomarker feedback; SBF = susceptibility biomarker feedback.

Impact of treatment on smoking behavior

Chi-square tests were performed to evaluate between-groups differences in smoking behavior at 12 months post-treatment. As shown in Table 2, there was a significant effect of treatment on the quit attempt variable ($p = .05$). Eighty-five percent of those smokers in the SBF group reported trying to quit compared with 81% of smokers in the EBF group and 73% of smokers in the QSC group. Pairwise comparisons revealed that the significant difference was between the SBF and QSC groups. However, there were no significant overall or pairwise differences in 30-day quit rates. Overall, only 13% of study participants reported 30-day cessation.

A 3 (Group: QSC, EBF, SBF) × 3 (Time: Baseline, 2 Months, 12 Months) repeated measures analysis of variance was conducted to examine between-groups differences in changes in smoking rates across time. As shown in Figure 1, the results revealed a significant main effect for time, $F(2, 620) = 56.59, p = .0001$, but no significant Time × Treatment interaction, $F(4, 620) = 0.83, p = .50$. Despite an initial reduction in smoking rates at the 2-month follow-up, smoking rates increased in all groups by the 12-month follow-up.

Main and moderating effects of baseline stage and self-efficacy

Multivariate regression modeling was performed to test the main effects and moderating effects of baseline stage and baseline quitting self-efficacy. Controlling variables were identified through bivariate analyses. Any demographic or baseline smoking history variable with a significant association ($p < .10$) with a dependent variable was controlled in the multivariate model of that dependent variable. There were no significant associations between potential confounding variables and the two dichotomous dependent variables (quit attempt and quit for at least 30 days). For the logistic regression models, dummy variables for treatment effects (EBF vs. QSC and SBF vs. QSC) were entered on the first step. The moderator variables (baseline stage and self-efficacy) were entered on the second step, and the interaction terms (moderator variables by dummy variables) were entered on the third step.

The logistic regression model for quit attempt revealed a significant main effect for treatment. Participants in the SBF group were more than two times more likely to make a quit attempt than participants in the QSC group (odds ratio [OR] = 2.13, confidence interval [CI] = 1.13–4.01, $p = .02$). There was not a significant difference be-

Table 2. The impact of treatment quitting outcomes

	Treatment group							
	QSC ($n = 137$)		EBF ($n = 156$)		SBF ($n = 133$)			
Dependent variable	*N*	%	*N*	%	*N*	%	χ^2	*p*
Quit attempt								
Yes	101	73.2	127	81.4	113	85.0	6.2	.05
No	36	26.8	29	18.6	20	15.0		
30-day quit								
Smoking	115	83.3	136	87.2	118	89.4	2.2	.33
Abstinent	23	16.7	20	12.8	14	10.6		

Note. QSC = quit-smoking counseling; EBF = exposure biomarker feedback; SBF = susceptibility biomarker feedback.

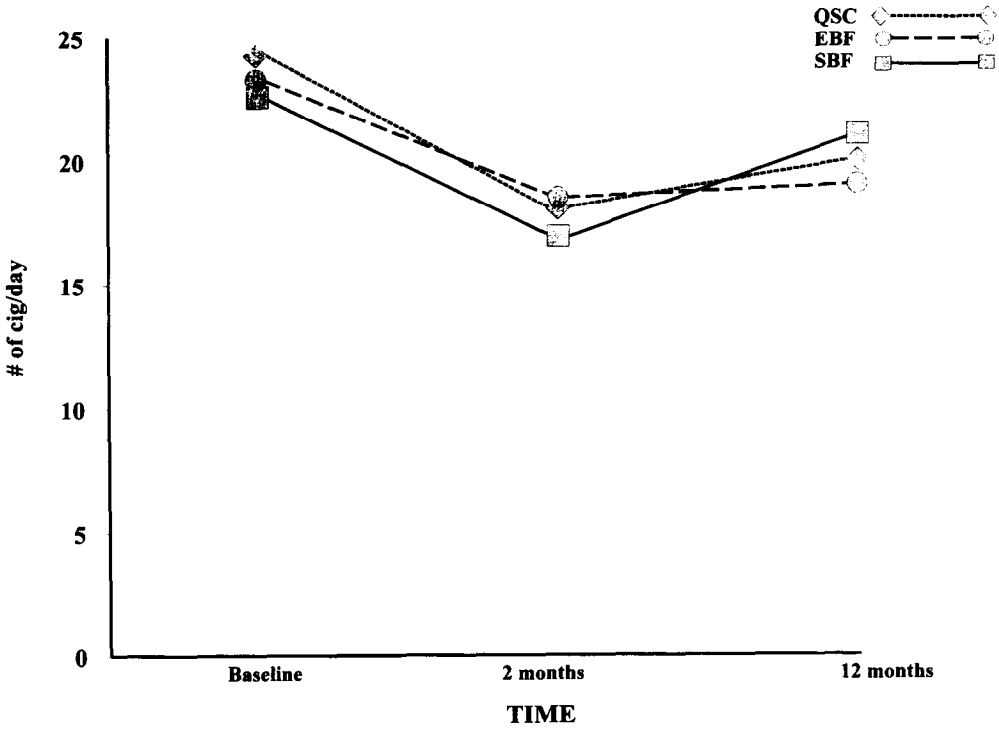

Fig. 1. Changes in smoking rate over time.

tween EBF and QSC participants (OR = 1.57, CI = 0.89–2.78, p = .12). The results also revealed a significant effect for baseline stage of change (OR = 3.22, CI = 1.77–5.85, p = .0001). The OR suggested that those smokers in the preparation stage at baseline were more than three times more likely to make a quit attempt over the 12 months following treatment. The interactions between treatment and the moderator variables were dropped from the model because they were not significant. Therefore, neither stage of change nor baseline self-efficacy moderated the relationship between treatment and quit attempts.

The logistic regression model for 30-day cessation revealed no significant main effects for treatment (OR = 0.57, CI = 0.28–1.17, p = .13 for SBF vs. QSC; OR = 0.73, CI = 0.38–1.40, p = .34 for EBS vs. QSC) or baseline stage of change (OR = 1.47, CI = 0.83–2.61, p = .18) or self-efficacy (OR = 1.16, CI = 0.65–2.08, p = .69), and there were no significant interactions. Therefore, neither stage of change nor baseline self-efficacy moderated the efficacy of treatment on cessation.

Among participants who reported continued smoking at the 12-month follow-up, a linear regression analysis was performed to examine the effects of treatment on follow-up smoking rate. In bivariate analysis, gender, marital status, and ethnicity were associated significantly with smoking rate: $t(234) = 1.84, p = .03$; $t(350) = -2.04, p = .04$; and $t(93) = 4.23, p = .01$, respectively. Thus, gender, marital status, and ethnicity were controlled in the linear regression model. This analysis did not reveal any significant main or interactive effects of treatment, $F(1, 335) = 0.45, p = .50$, for SBF versus QSC, and $F(1, 335) = 0.23, p = .63$, for EBF versus QSC, and stage or change, $F(1,335) = 1.43, p = .23$, and self-efficacy, $F(1, 335) = 1.33, p = .25$.

Impact of treatment on depression

A 3 (Group: QSC, EBF, SBF) × 3 (Time: Baseline, 2 Months, 12 Months) repeated measures analysis of variance was performed to examine the effects of treatment on depression across time. The results revealed a significant main effect for time, $F(2, 818) = 3.67, p = .03$, but no Treatment or Time × Treatment interaction, $F(4, 818) = 1.40, p = .23$. Thus, the initial increase in depression in the genetic susceptibility group was not maintained over time (see Fig. 2).

DISCUSSION

This article extends our previous report (Lerman et al., 1997) by describing the 1-year outcomes of a randomized trial of genetic susceptibility feedback incorporated into a minimal contact smoking-cessation treatment. Earlier, we reported that genetic feedback had significant positive effects on quitting-related beliefs and motivations; however, there was no impact on 2-month cessation rates. The results of the current analysis are consistent with this earlier analysis in that we found evidence for a significant impact of genetic feedback on the likelihood of a quit attempt but no effect on actual cessation at the 12-month follow-up.

Thus, the short-term and long-term results of this randomized trial indicate that, although genetic susceptibility feedback has the intended effects on motivation to quit, this approach is not likely to promote cessation when delivered in conjunction with a minimal contact treatment approach. The smokers who were more likely to participate in our program tended to smoke more and for a greater number of years than

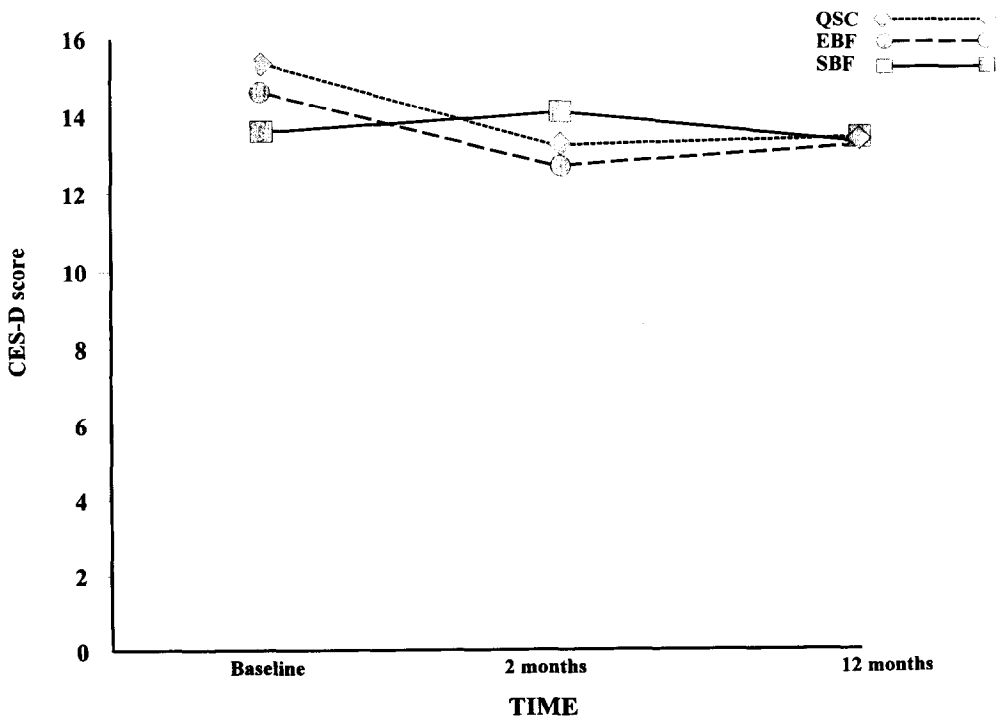

Fig. 2. Changes in depression over time.

those smokers who did not participate. Minimal contact approaches may not be intensive enough for more dependent smokers (Hughes & Glaser, 1993). More intensive smoking-cessation treatment may be needed for the heightened motivation to translate into successful quitting. For example, recent studies have shown that the addition of follow-up telephone counseling can boost cessation rates by as much as 50% (Orleans et al., 1991). In addition, when supplemented with nicotine replacement therapy, the effectiveness of minimal contact approaches is also enhanced (Fiore, Smith, Jorenby, & Baker, 1994; Leischow, Hill, & Cook, 1996; Silagy, Mant, Fowler, & Lodge, 1994). These strategies to address the nicotine addiction directly and to impart additional skills and support may provide a more fruitful context for the evaluation of genetic susceptibility feedback.

In this study, we also evaluated two potential moderators of the impact of genetic susceptibility feedback on cessation, stage of readiness to quit smoking, and self-efficacy. Stage of readiness to quit smoking was found to be a significant predictor of making a quit attempt, independent of treatment group assignment; 90% of smokers in the preparation stage at baseline had made a quit attempt at the 12-month follow-up, compared with 75% of smokers in the precontemplation/contemplation stage. Stage of change did not predict actual changes in smoking practices, however. Contrary to previous reports (Brandon et al., 1990; DiClemente et al., 1985), self-efficacy regarding quitting did not predict any of the smoking outcomes. It is possible that the one-item self-efficacy measure was not sensitive enough to detect associations between self-efficacy and the smoking outcomes (Garvey et al., 1992; Nides et al., 1995). Neither stage of readiness nor self-efficacy were found to moderate the impact of treatment. The absence of significant moderation by these variables is consistent with the interpretation that the failure of genetic feedback to promote cessation was not related to a motivational deficit but rather to the inability to overcome the addiction.

On a more positive note, the present analysis indicated that the initial increases in depressive symptoms observed at the 2-month follow-up in the genetic susceptibility feedback group (Lerman et al., 1997) were not sustained at the 12-month follow-up. The promotion of psychological distress following notification of an increased genetic risk is a particular concern (Croyle, Achilles, & Lerman, 1997). Psychological distress may be generated especially when the behaviors required to reduce risk (i.e., quitting smoking) are very difficult to control. Because of the possibility of short-term adverse psychological effects of genetic testing, future studies incorporating such testing in smoking-cessation treatment should provide adequate psychological support to those participants given an increased risk assignment.

One potential limitation of the present investigation is that we relied on self-report of smoking status at the 12-month follow-up. There is considerable debate, however, over the utility of biochemical verification of smoking status after cessation treatment, especially in the context of a minimal contact intervention (Glasgow et al., 1993; Wilson, Wallston, King, Smith, & Heim, 1993). In addition, only 13% of the sample reported being abstinent at the 12-month follow-up. Also, none of the significant findings related to the abstinence variable.

In conclusion, the present investigation found that genetic susceptibility feedback has a significant impact on the likelihood of a quit attempt. It is important to note, however, that it would be premature to provide genetic testing for lung cancer susceptibility in a clinical smoking practice. More research needs to be conducted on the genetics of smoking and lung cancer and on how smokers in general and different subpopulations of smokers respond to genetic susceptibility feedback. These types of

studies will provide information to assist in the development of guidelines for use in clinical smoking practice. Future research may want to evaluate the impact of genetic susceptibility feedback as a component of a more intensive smoking-cessation treatment program.

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